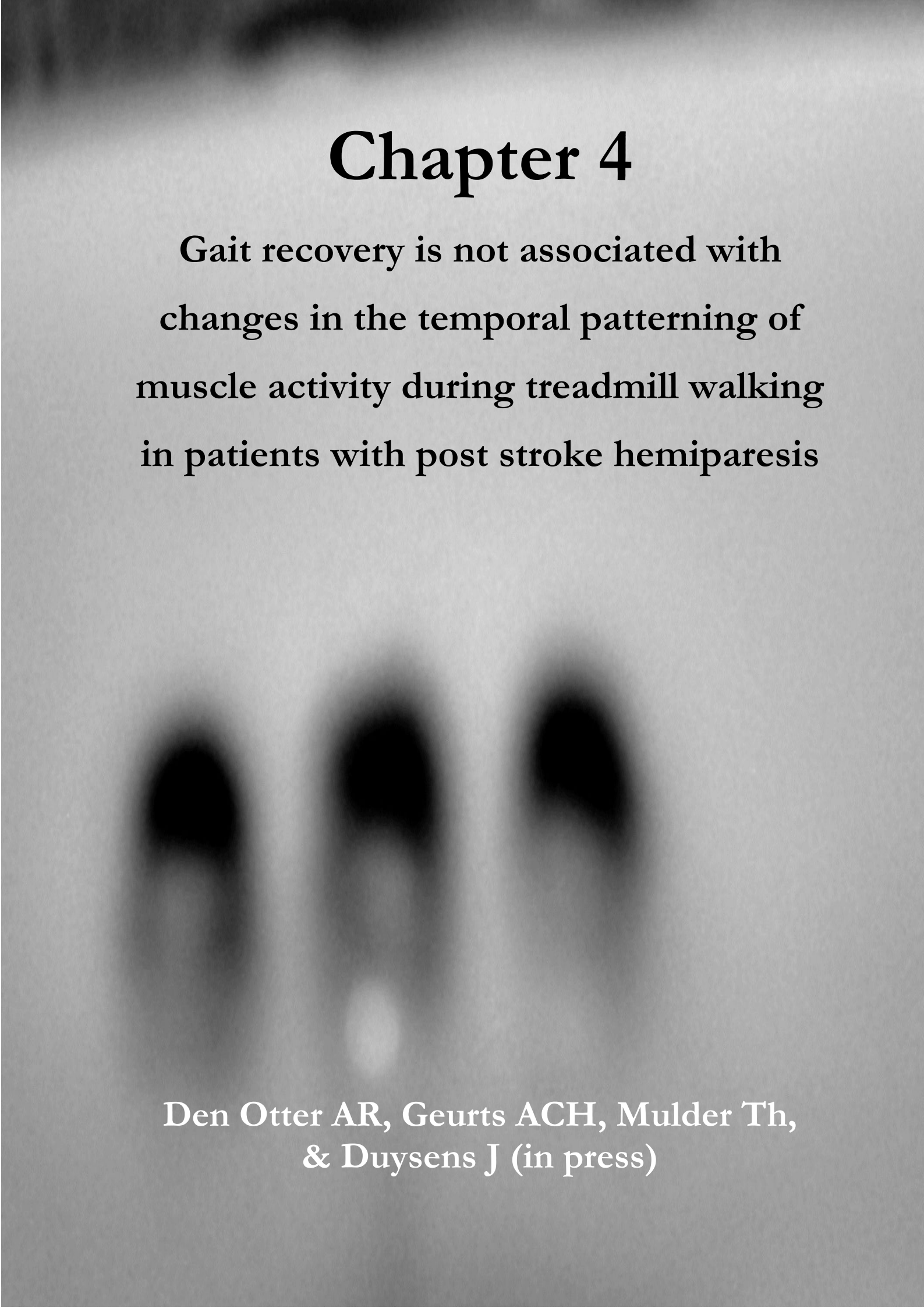
Gait control after stroke

Otter, Arie Robert den

Chapter 4

Gait recovery is not associated with changes in the temporal patterning of muscle activity during treadmill walking in patients with post stroke hemiparesis



Den Otter AR, Geurts ACH, Mulder Th, & Duysens J (in press)

Abstract

The objective of this study was to establish whether functional recovery of gait in patients with post-stroke hemiparesis coincides with changes in the temporal patterning of lower extremity muscle activity and coactivity during treadmill walking. Electromyographic (EMG) data from both legs, maximum walking speed, the amount of swing phase asymmetry and clinical measures were obtained from a group of post-acute patients with hemiparesis, as early as possible after admission in a rehabilitation centre (mean time post stroke 35 days) and 1, 3, 6, and 10 weeks later, while all patients participated in a regular rehabilitation program. EMG data from the first assessment were compared to those obtained from a group of healthy controls to identify abnormalities in the temporal patterning of muscle activity. Within subject comparisons of patient data were made over time to investigate whether functional gait recovery was accompanied by changes in the temporal patterns muscle (co-) activity. EMG patterns during the first assessment showed a number of abnormalities on the paretic side, namely abnormally long durations of activity in biceps femoris (BF) during the single support (SS) phase and in gastrocnemius medialis (GM) during the first double support phase (DS1). Furthermore, in both legs a prolongation of the activity was seen in the rectus femoris (RF) during the SS phase. In addition, the duration of BF-RF coactivation was longer on the paretic side than it was in controls. Over time, the level of ambulatory independence, body mobility, and maximum walking speed increased significantly, indicating that substantial improvements in gait ability occurred. Despite these improvements, durations of muscle (co-) activity and the level of swing phase asymmetry did not change during rehabilitation. More specifically, timing abnormalities in muscle (co-)activity that were found during the first assessment did not change significantly, indicating that these aberrations were not an impediment for functional gait improvements.

The main conclusion is that normalization of the temporal patterning of gait related muscle activity in the lower extremities is not a prerequisite for functional recovery of gait in patients with post stroke hemiparesis. Apparently, physiological processes other than improved temporal muscular coordination must be important determinants of the restoration of ambulatory capacity after stroke.

Introduction

During human walking, the muscle activity of the lower extremities needs to be well coordinated to provide support, dynamic balance, propulsion, and foot clearance. These gait tasks are temporally coupled to particular phases of the gait cycle and, as a result, the timing of lower extremity muscle activity during healthy human walking follows characteristic patterns. After unilateral supratentorial stroke, the neuromuscular control of walking may undergo radical changes which include muscle weakness on the body side contralateral to the lesion, and often dramatic changes in the temporal organisation of muscle activity. Several electromyographic (EMG) studies have been conducted to obtain information on the patterning of lower extremity muscle activity in hemiparetic gait. Despite large interindividual differences among patients, some common timing abnormalities in muscle activity patterns have been reported. Among these deviations are the premature activity of the paretic calf muscles during the terminal swing and initial stance phase (Perry et al. 1978; Lamontagne et al. 2001; Burridge et al. 2001), the prolonged stance activity of hamstrings and quadriceps muscles on both the paretic (Hirschberg and Nathanson, 1952; Peat et al., 1976; Knutson and Richards, 1979; Shiavi et al., 1987) and the nonparetic side (Wortis et al., 1951; Shiavi et al., 1987), and the absence of activity in the paretic ankle dorsiflexors during the late swing and early stance phase (Burridge et al., 2001; Perry, 1993). These abnormalities in the patterning of lower extremity muscle activity determine, to a substantial degree, the quality and the functional limitations of the hemiparetic gait pattern. Therefore, the development of effective treatment strategies for gait impairments following stroke requires insight into these neuromuscular timing abnormalities, how they change over the course of gait recovery, and how they can be modified.

Although alterations in the timing control of gait related muscle activity may impair gait ability, and therefore forms a potential target for gait rehabilitation, there is as yet little evidence available to show that gait recovery is indeed associated with changes in muscle activation patterns during walking. To provide an empirical basis for the development of gait training strategies, it is necessary to understand which aspects of muscle control remain stable over the course of gait recovery, and what aspects are susceptible to change. For this purpose, it does not suffice to focus solely on the identification of abnormalities in muscle control using cross-sectional measurements. It is also necessary to explore the changes on such aberrations in a longitudinal study, where repeated measurements are made in the same patients over the course of gait recovery. To our knowledge, only one EMG

study has been conducted that has addressed changes in muscle activity over the course of recovery in patients with stroke. Shiavi and co-workers (1987) classified 12 patients according to the type of muscle synergy apparent in the EMG patterns (cf. Knutson and Richards, 1979), and found that synergy patterns changed over a period of approximately 1 year. However, identification of synergy patterns and changes over time was made on the basis of visual inspection of the ensemble averaged EMG patterns. Furthermore, because walking speed affects the amplitude of muscle activity, and also the timing of activity of specific muscle groups (Hof et al., 2002; den Otter et al., 2004), it is not clear whether differences in gait speed between both assessments may have affected the results. A clearer view of recovery related changes in muscle activity after stroke may arise when the identification of abnormality and change is based on objective statistical criteria, and when the influences of walking speed are ruled out.

Hence, the primary goal of the present study was to study changes in the temporal structure of lower extremity muscle activity during the recovery of gait ability in hemiparetic stroke patients, using objective statistical criteria and controlling for walking speed. In addition to the analysis of the temporal patterning of individual muscles, this study focuses on the assessment of patterns of coactivity between antagonistic muscle pairs. Investigation of coactivation may provide valuable information on neuromuscular coordination in hemiparetic gait, because altered coactivation levels may be indicative of pathological muscle synergies (Knutsson and Richards, 1979; Shiavi et al, 1987), or compensatory coordinative strategies which may be different for the paretic and the nonparetic side (Lamontagne et al., 2000). Because gait recovery may be associated with the development of more selective muscle control or with the emergence of compensatory coactivation strategies, a secondary goal was to study the evolution of muscle coactivity in patients with hemiparetic stroke during their regular, post-acute rehabilitation.

Methods

Subjects

A convenience sample of 14 rehabilitation inpatients with stroke, who were consecutively selected based on inclusion and exclusion criteria and their willingness to cooperate, participated in this study (8 females, 6 males). The mean age in this group was 54.7 yrs (sd=9.9; range 39 – 71 yrs). The mean time since stroke onset at the time of inclusion was 35 days (sd= 7.69), and ranged from 23 to

52 days. All patients suffered an ischemic stroke; five of the patients had suffered a infarction in their left hemisphere , whereas 9 patients had suffered an infarction in their right hemisphere; To be included, patients were required to (1)-. have suffered a first-ever supratentorial stroke (due to either haemorrhage or infarction), (2)-. have been admitted to the rehabilitation centre with an aim of restoring independent gait, and (3)-. have a Functional Ambulation Categories score of at least 2 (Patient needs continuous or intermittent support of one person to help with balance or coordination) or at the most 4 (Patient can walk independently on level ground, but requires help on stairs, slopes or uneven surfaces). Exclusion criteria were: (1) severe forms of aphasia or other cognitive problems that could hinder communication or cooperation (2). severe affective or psychiatric impairments; (3) visuospatial neglect, as indicated by abnormal scores on two or more of the following tests: the line bisection test (Schenkenberg et al. 1980), the letter cancellation task (Diller et al. 1974), the Bells test (Gauthier et al., 1989), and the clock drawing test (Wilson et al., 1987); and (4) a history of orthopaedic or neurological (other than stroke) disorders.

All patients underwent a clinical examination by an experienced physician, providing data for lower limb motor selectivity, sensation modalities, and trunk control (cf. de Haart et al., 2004). Lower limb motor selectivity was scored according to the six motor stages as defined by Brunnstrom (1966). At the time of inclusion, the Brunnstrom scores in the patient group ranged from 3 (increased muscle tone with active movements mainly in rigid extension synergy) to 5 (muscle tone normalization with some degree of selective muscle control). The lower limb sensation modalities were assessed by testing position sense at the affected ankle joint in 3 different positions of dorsi- and plantarflexor by mirroring with the nonparetic ankle. In this test, the patient was supine, and the paretic leg was slightly lifted by the physician. A score was recorded as 'impaired' if the patient had a mirroring error greater than 1. In the group of 14 patients, 6 patients had impaired sensation modalities at the time of inclusion. The patient's trunk control score was determined by the sitting balance item of the Trunk Control Test (Wade, 1992). Control was rated as 'impaired' if the patient was unable to stay up sitting on the edge of a bed, feet off the ground, for 30 seconds. At the time of inclusion, 11 of the 14 patients that participated in this study had impaired trunk control (see table 1 for individual patient characteristics).

All patients received between 5 and 7 sessions of physiotherapy per week., with an approximated duration of 30 minutes per session. In addition, all patients

received three 30 minute sessions of occupational therapy per week. These individual therapies were augmented by small-group therapy for improving gross motor skills. The group therapy occupied at least 60 minutes of each working day. This motor rehabilitation was embedded in a more extensive, individualized neurodevelopmental oriented rehabilitation program, in which much emphasis was put on optimal use of the paretic body side.

Gait data of the patient group were compared with data obtained from 14 healthy controls. The control group consisted of 8 females and 6 males, with a mean age of 42.8 years (sd=12.3). All control subjects did not suffer from any neurological or orthopaedic disorder. All participants gave their written informed consent before study entry. The study was approved by the regional medical-ethical committee Arnhem-Nijmegen.

Procedure

Patients were assessed on 5 occasions during their rehabilitation process. The first assessment took place as early as possible after admission. Subsequent assessments took place 1, 3, 6, and 10 weeks after the first assessment. At each assessment, scores were obtained for the Rivermead Mobility Index (RMI) (Collen et al. 1991) and for the level of ambulatory independence using the Functional Ambulation Categories (FAC) (Holden et al., 1984). In addition, the maximum walking speed on a treadmill that could be maintained for 40 seconds was registered. Finally, at each assessment, spatiotemporal gait parameters and electromyographic data were obtained using instrumented gait analysis. All clinical tests were performed by the primary investigators (RdO) or the patient's therapist, but the evaluator was the same throughout the assessment, for each patient. All gait assessments were performed by the primary investigator, assisted by one physiotherapist.

During gait analysis, subjects walked on a motor-driven treadmill (walking surface 200 x 70 cm), for 40 seconds. During the first gait assessment, patients were encouraged to walk at the maximum treadmill speed they could maintain for a period of 40 seconds. During subsequent gait analyses (assessments 2 to 5), patients were tested at the same speed in order to allow valid comparisons. Subsequently, the treadmill speed was increased until the maximum speed was reached that could be maintained for 40 seconds. This speed served as an indication of the level of ambulatory skill at the time of the assessment. In the control group, half of the subjects walked at a treadmill speed of 0.28 ms^{-1}, and the other half walked at 0.56 ms^{-1}, so that the mean gait speed in the control group (0.42 ms^{-1}) matches the mean

gait speed in the patient group (0.37 ms^{-1}) rather well. It is quite unlikely that this minor difference in the mean gait speed between the two groups is a main confounder in this study, especially in the light of other literature showing that substantial differences (> 0.56 ms^{-1}) in gait speed are required to induce differences in timing characteristics in EMG (e.g. Hof et. al, 2002; den Otter et al., 2004).
When, at a particular assessment, a patient was still unable to walk independently on the treadmill, this subject was allowed to receive occasional manual assistance by one physiotherapist to maintain balance. Also, in case of sudden disturbances of postural balance patients were allowed to grasp a rail in front of them. This was done in order to enhance safety and to reduce the potential effects of fear which may result in enhanced co-activation levels, (Maki, 1991; Adkin et al., 2000). However, sustained contact with the rail was not tolerated. All patients wore a harness to enhance safety, but no body weight support was supplied.

Data recording
EMG recordings were made using disposable surface electrodes (MediTrace ECG 1801 Pellet, (Ag/ AgCl)) with a 10-12 mm diameter and an interelectrode distance of 24 mm (Graphics Controls, Buffalo NY, USA). Four lower extremity muscles were recorded from both legs: biceps femoris (BF), rectus femoris (RF), gastrocnemius medialis (MG), and tibialis anterior (TA). Electrode placement was based on SENIAM recommendations (Freriks et al. 1999), according to which the sensors were placed relative to specified anatomical landmarks. These specifications enabled us to place the electrodes on identical skin locations throughout the series of assessments. The EMG signals were checked for crosstalk by performing standard muscle tests in a seated position.

The incoming electromyographic signals were fed to a K-lab SPA 20/8 pre-amplifier with a common mode rejection ratio > 95 db and a noise level of < 1 μV rms. Subsequently, signals were high pass filtered using a third order Butterworth filter (- 3db point at 20 Hz), and low pass filtered by means of a second order Butterworth filter (- 3db point at 500 Hz). The EMG signals were digitised at 2400 Hz and stored on a computer hard disk for offline processing. Light reflective markers were attached to the heel of each foot, and 3-D displacements were recorded by 5 infrared cameras (PRIMAS™). These signals were then digitized at 100 Hz.

Data analysis

In order to determine the relative duration of muscle activity for different phases of the gait cycle, EMG signals were dichotomized into periods of activity and inactivity. During offline processing, EMG data were first high pass filtered at 10 Hz (to attenuate movement artifacts), and full wave rectified and then low pass filtered at 25 Hz. Next, all individual datapoints in the EMG linear envelopes were partitioned into subsets of data using a k-means clustering algorithm. The goal of k-means clustering is to find similarities between data points and to group these data points according to their similarities (Mac Queen, 1967). By definition, periods of muscle inactivity in the rectified and low pass filtered signal form a relatively homogeneous subset of data, so that signals can be dichotomized by assuming that data belonging to the cluster with the lowest mean value correspond with periods of muscle inactivity, and data belonging to other clusters to periods of muscle activity. For the data analyzed in this study, the number of clusters was set, a priori, to 5. The method described here allows for detection of short, transient bursts of muscle activity, and does not require the setting of a priori thresholds.

In order to assess the reliability of the results obtained from this detection method, EMG data (BF, RF, TA, and GM of the right leg) were collected during two separate trials in a group of 9 healthy young adults (five females, four males; mean (S.D.) age 22.4 (2.35) years; height 1.81 (6.7) m; body mass 66.6 (7.84) kg) during 40 seconds of treadmill walking at a speed of 0.83ms^{-1}. For each individual subject the difference in the duration of detected activity for DS1, SS, DS2, and SW were calculated between both trials, and subsequently the median difference over the group was calculated. In addition, Spearman's correlation coefficient was calculated for the group. The results are shown in table 2. The median percentage difference between the two trials ranged from 1.2 % (BF during DS1) to 9.2% (BF during DS2), indicating that the duration of activity was detected with good reproducibility between trials. For the group, the correlation coefficients were generally good to very good, ranging from 0.65 (BF during SS) to 1.00 (BF during DS1).

Speed distribution analysis of the heel marker data (Peham et al., 1999) was used to determine heel strike and toe off for both legs. Based on this information, the swing phase, the first and second double support phase, and the single support phase, were determined for each leg (for an explanation of these gait phases, and an example of the EMG data with the phases of muscle activity detected by the clustering algorithm, see figure 1). The number of strides that were used for

analysis differed slightly between subjects and between assessments (e.g. due to interindividual differences in gait speed). The minimum number of strides that was analyzed for one assessment was15, and the maximum difference between assessments within one patient was 5 strides. In order to obtain an estimate of the

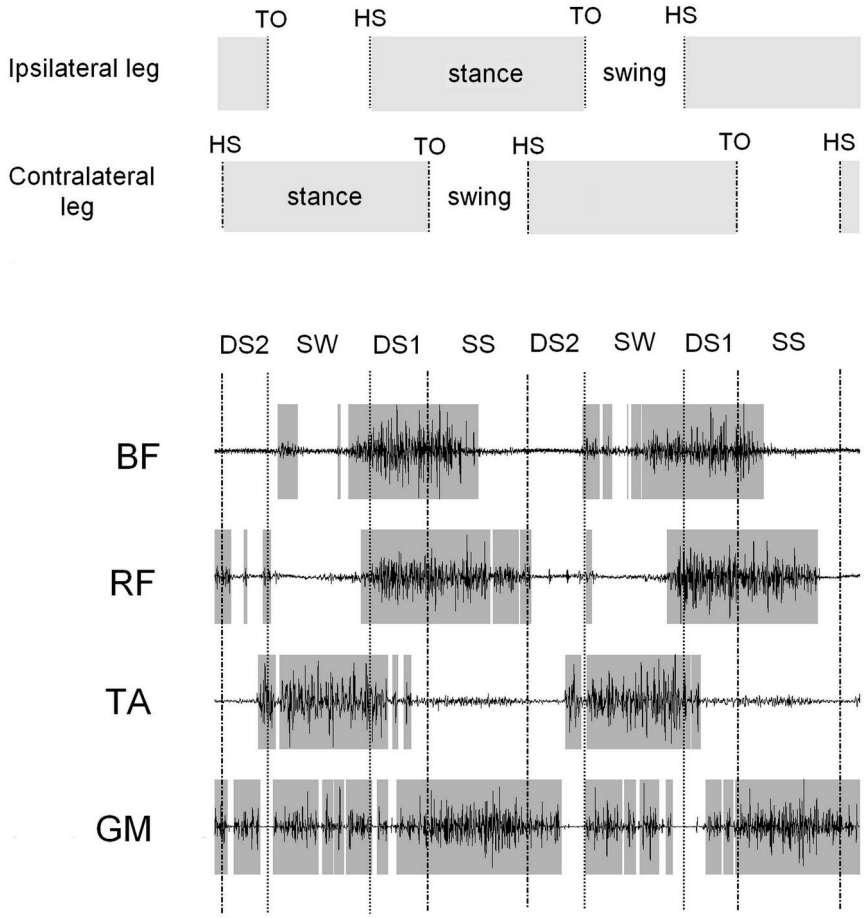


Figure 1. An example of the gait phases (stance and swing phases, top part of the figure) and the corresponding DS1, SS, DS1, and SW phases of the ipsilateral leg, superimposed on the raw EMG signal. Periods in the EMG signals that were classified as 'activity' by the clustering algorithm are indicated by gray shaded areas.

temporal asymmetry between the paretic and the nonparetic leg in patients, swing phase asymmetry was calculated as follows:

$$\frac{\text{duration of swing phase paretic leg} - \text{duration of swing phase nonparetic leg}}{0.5 * (\text{duration of swing phase nonparetic leg} + \text{duration of swing phase paretic leg})}$$

For each subject, and for all of the gait phases, the percentage of time during which a muscle was considered active was calculated for each step. Subsequently, these

percentages were averaged over all gait cycles, for each subject. Based on the dichotomised signals, the relative amount of time that two muscles were simultaneously active was calculated to obtain an estimate of the duration of coactivation between these muscles. This way, the relative duration of coactivation was calculated between BF and RF, and between TA and GM, for both legs. All offline analyses of EMG signals and marker data were done using custom software made in Matlab™.

Statistical analysis

In order to identify abnormalities in the EMG patterns of the patients, the durations of muscle activity and coactivity obtained on the first gait assessment were compared to data obtained from the control group. A Wilcoxon rank-sum ranks test was performed to test for differences between patients and controls.

Within the patient group, time related changes in RMI scores, FAC scores, maximum walking speed, swing phase asymmetry and relative durations of muscle activity and coactivity (within subjects factor TIME) were tested non-parametrically with the Friedman test. In the case of a significant overall TIME effect, post hoc analysis was performed to test for differences between particular assessments, using a Wilcoxon matched pairs – signed ranks test. In this case, Bonferoni corrections were made to maintain the familywise alpha level at .05.

Results

Subjects

All of the 14 patients were able to participate in at least 4 of the 5 assessments. Four patients were unable to participate in the 5th and final assessment because they were unwilling to return to the clinic after discharge. In addition, for 1 subject, gait data of the final assessment were lost due to computer failure. In order to utilize as much of the collected data as possible, and because data from the 5th assessment may provide information on possible changes in EMG patterns that occur later during recovery (> 6weeks), the data of the 4th and the 5th assessment were averaged when data were available for all 5 assessments. In this way, clinical, spatiotemporal, and electromyographic data could be compared between 4 moments in time, for all 14 patients. Data from the 5th assessment were included because they may provide information on possible changes in EMG patterns later during recovery (> 6weeks).

The mean time post stroke at the first assessment was 35 days (sd=7.7), 44

days (sd=7.0) at the second assessment, 60 days (sd=8.01) at the third assessment, and 90 days (sd=9.4) at the fourth assessment.

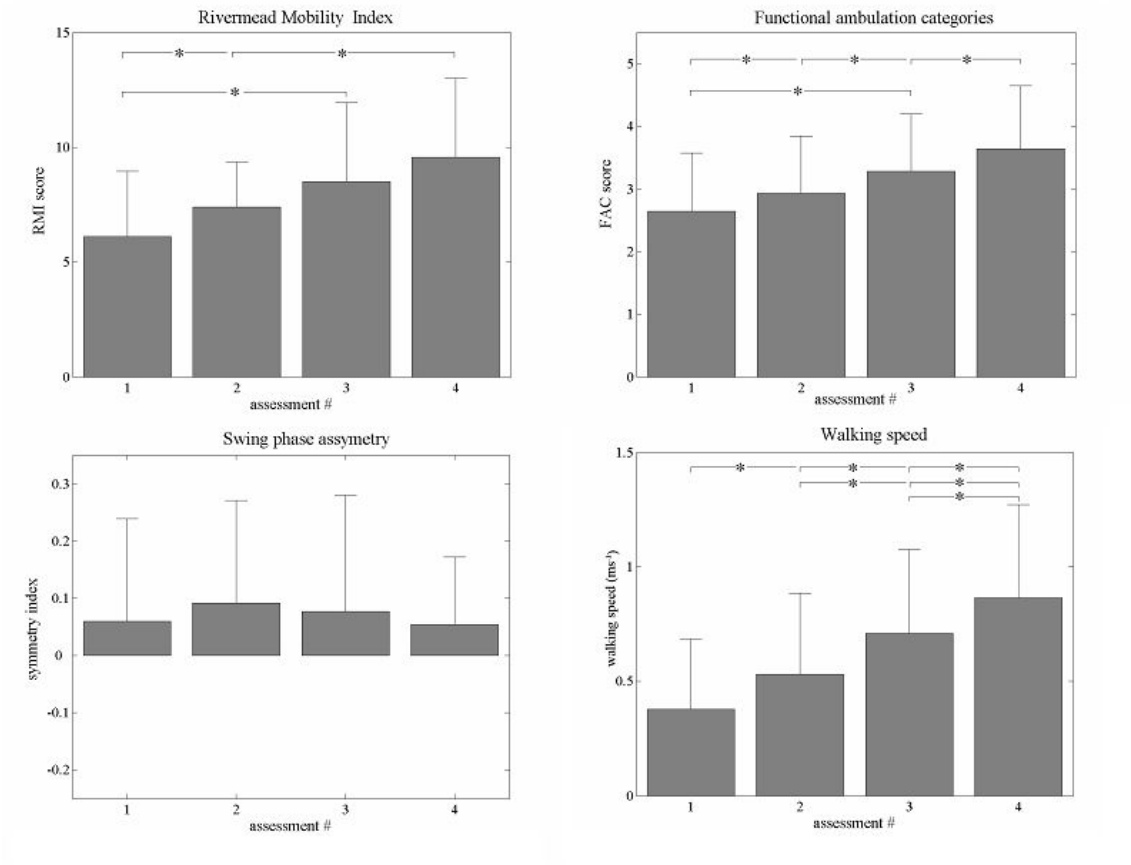


Figure 2. Means and SDs of the patient group for the Rivermead Mobility Index (top left panel), the Functional Ambulation Categories (top right panel), asymmetry of swing-phase duration (bottom left panel), and maximum walking speed (bottom right panel) at all 4 assessments. (* = statistically significant at $p < .05$)

Clinical measures of mobility and ambulatory independence

The mean and standard deviations of the RMI scores at all 4 assessments are depicted in the top left panel of figure 2. The RMI scores showed that the general mobility of patients increased significantly over time (Chi^2 (df=3)= 11.85; $p<.05$). Post hoc comparisons between the different assessments revealed that significant changes in RMI occurred between assessments 1 and 3 ($Z=-2.30$; $p<.05$), between assessments 1 and 4 ($Z=-2.23$; $p<.05$), and between assessments 2 and 4 ($Z=-1.98$; $p<.05$).

The mean FAC scores (+sd's) are presented in the top right panel of figure 2, top right panel). The observed increases in ambulatory independence resulted in a statistically significant time effect (Chi^2 (df=3)=11.97; $p<.05$). Post hoc analysis showed that FAC scores were significantly different between assessments 1 and 3

(Z=-2.00; p<.05), and that the scores at assessments 1 (mean=2.64; sd=0.93), 2 (mean=2.93; sd=0.92) and 3 (mean=3.29; sd=0.91) were all significantly different from the FAC scores at assessment 4 (mean=3.64, sd=1.01; p<.05).

Spatiotemporal measures

The mean maximum walking speeds on the treadmill at all 4 assessments are depicted in the bottom right panel of figure 2, and show a clear increase over time (Chi^2=41.19; p<.001). The mean walking speed for the patient group at the first assessment was 0.37 ms^{-1} (sd=.31), and ranged from 0.11 to 1.06 ms^{-1}. At the last assessment, the mean maximum walking speed was 0.87 ms^{-1} (sd=0.41), with individual speeds ranging from .28 to 1.44 ms^{-1}.

At all 4 assessments, positive but relatively low degrees of swing-phase asymmetry were found, indicating that the mean duration was longer on the paretic side than on the nonparetic side. Statistical testing failed to show a time effect, indicating that the asymmetry in swing phase duration was stable over the course of time (Chi^2(df=3)=1.06; n.s.).

Temporal characteristics of muscle activity at the first assessment

Figure 3 shows the mean percentage of the duration of activity at the first assessment, for each muscle, and for each subphase of the gait cycle. In the BF of the paretic leg, an increased duration of muscle activity was found during the SS phase (73 %, sd=28%), when compared to controls (48%, sd=18%; Z=-2.67; p<.05).

The mean relative duration of BF activity during the SS phase in the nonparetic leg was also increased compared to controls (62.%, sd=23%), but this result did not reach significance (Z=-1.84; P=.069). As was the case for BF, the percentage of RF activity during the SS phase was higher in patients than in controls (58.%; sd=33%), in the paretic (81%, sd=29%; Z=-2.76,p<.05) as well as in the nonparetic leg (72.%, sd=31%. Z=-2.10, p<.05).

There were no statistically significant differences in TA activity between controls and patients for any of the four gait phases, neither in the paretic nor in the nonparetic leg. With respect to the paretic GM, the mean duration of activity during the DS1 phase was 58% (sd=22%), which was significantly longer than in controls (40%; sd=23%; Z=-2.068, p<.05)), indicating that in the paretic leg, GM activity started earlier. A similar difference could not be observed in the nonparetic

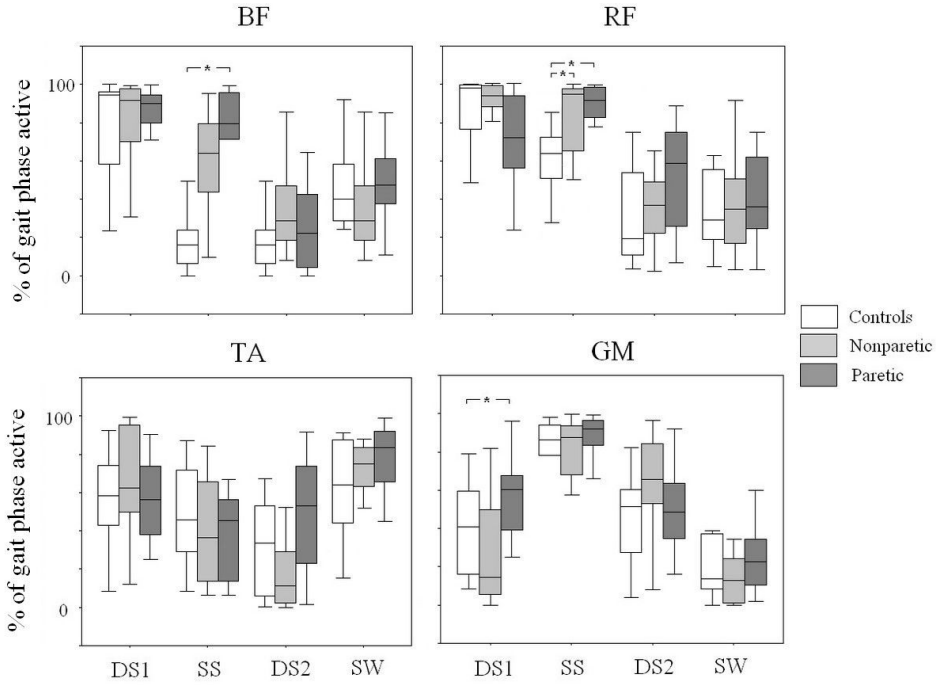


Figure 3. Boxplots (containing the median value, the minimum and maximum value, and values for the first and third quartile) of the percentage of the gait cycle phase during which a muscle was active at the first assessment in patients and in control subjects; (DS1 = first double support phase, SS = single support phase, DS2 = second double support phase, SW = swing phase; BF = Biceps femoris, RF = Rectus femoris, TA = Tibialis anterior, GM = Gastrocnemius medialis). The mean walking speed in the control group was 0.42 ms^{-1}

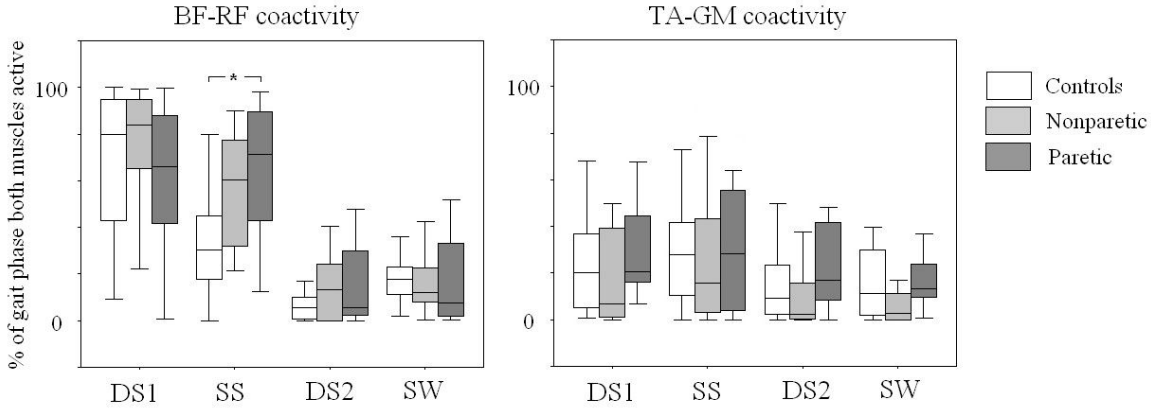


Figure 4. Boxplots (containing the median value, the minimum and maximum value, and values for the first and third quartile) of the percentage of the gait cycle phase during which two muscles were simultaneously active at the first assessment in patients and in control subjects (DS1 =first double support phase; SS= single support phase; DS2= second double support phase; SW=swing phase); (BF-RF= Biceps femoris – Rectus femoris; TA-GM= Tibialis anterior – Gastrocnemius medialis). The mean walking speed in the control group was 0.42 ms^{-1}

leg where GM activity showed an average duration of 27% (sd=26%) of the DS1 phase.

Relative timing of muscle activity: coactivation patterns

The relative durations of muscle coactivity during hemiparetic walking at the first assessment are depicted in figure 4. The amount of BF-RF coactivity during the SS phase was substantially higher in the paretic leg of the patients (63.%, sd=34%) than in the control group (31%, sd=21%) ($Z=-2.34$, $p<.05$). In the patients, as well as in the control subjects, the overall levels of TA-GM coactivity were generally low in both legs for all subphases of the gait cycle. There were no statistically significant differences between the two groups.

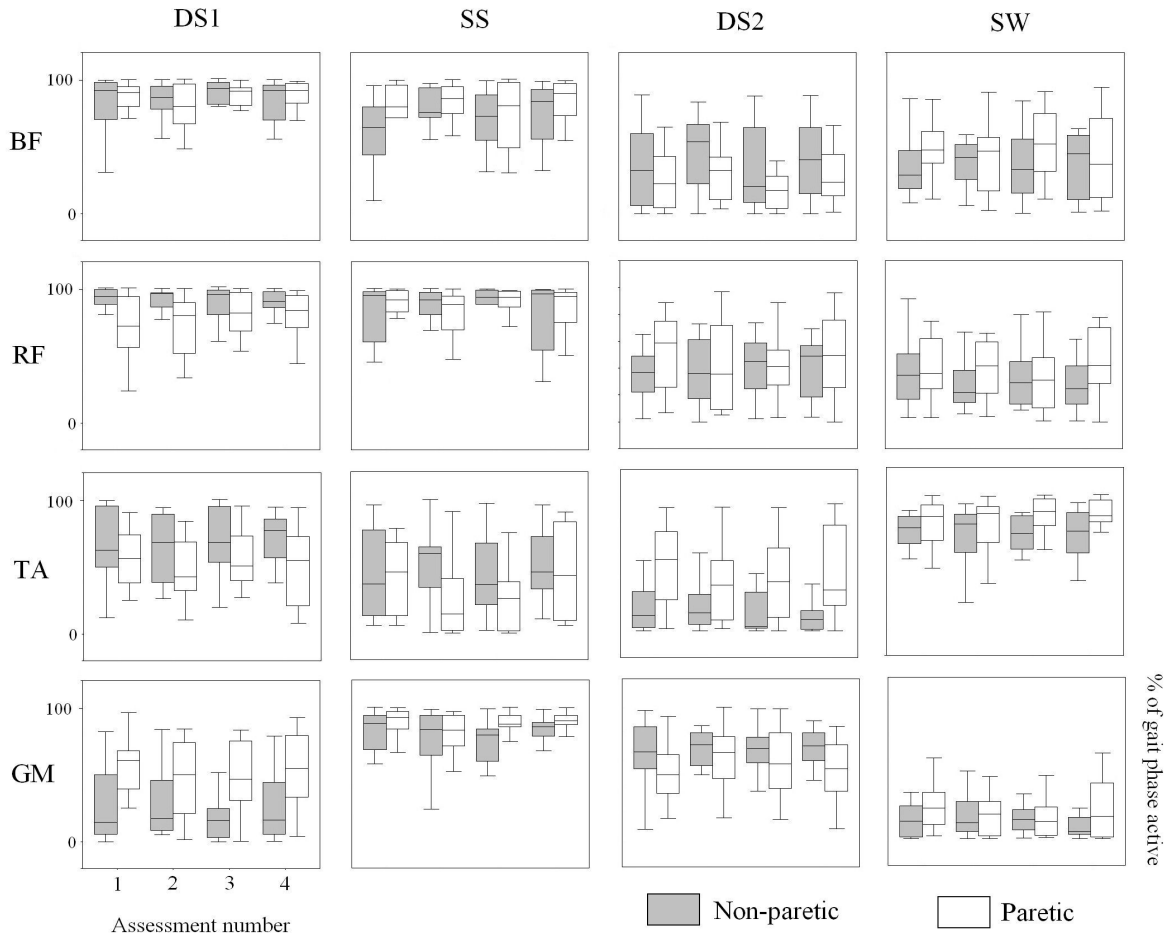


Figure 5. Boxplots (containing the median value, the minimum and maximum value, and values for the first and third quartile) of the percentage of the gait cycle phase during which a muscle was active at all 4 gait assessments (1-4) , for 4 phases of the gait cycle (DS1 =first double support phase; SS= single support phase; DS2= second double support phase; SW=swing phase; BF = Biceps femoris, RF = Rectus femoris, TA = Tibialis anterior, GM = Gastrocnemius medialis).

Patterns of muscle (co)activity during the course of gait recovery

Figure 5 shows the means and sd's of the relative durations of the activity of the BF, RF, TA and GM muscles in both the paretic and nonparetic leg at each of the 4 assessments, separately. For both legs, no statistically significant changes in the relative duration of activity were found for any of the 4 muscles, in any of the 4 gait phases that were studied.

The mean durations of agonist-antagonist coactivation in each of the 4 gait phases at all 4 assessments are depicted in figure 6. In the paretic as well as in the nonparetic leg, the durations of BF-RF and TA-GM coactivation did not change over time for any of the 4 gait phases. These results suggest that no systematic changes occurred in the duration of (co)activation between BF and RF, or between TA and GM, despite substantial improvements in the ambulatory status of the patient group.

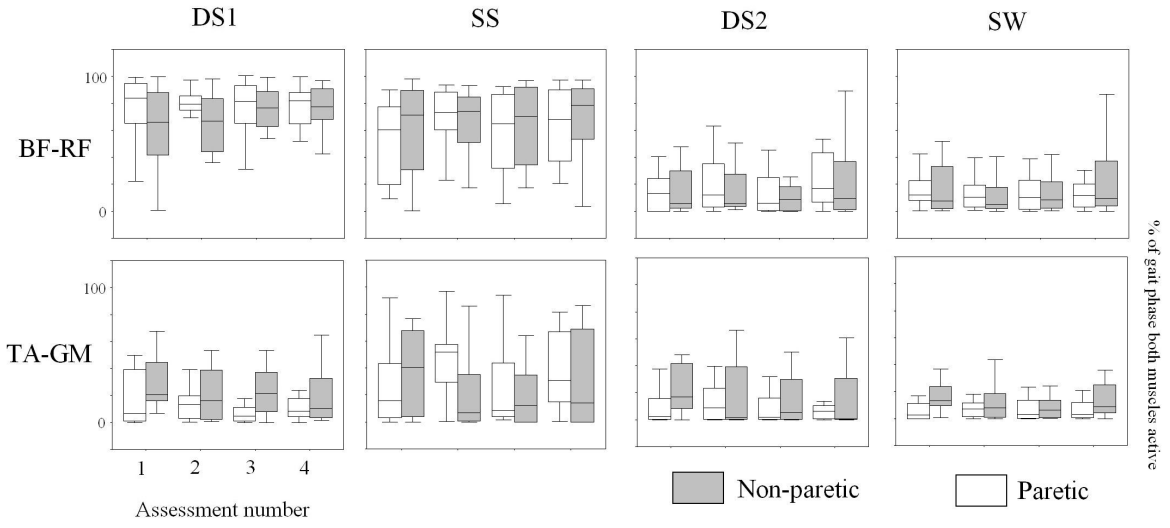


Figure 6. Boxplots (containing the median value, the minimum and maximum value, and values for the first and third quartile) of the percentage of the gait cycle phase during which two muscles were simultaneously active at all 4 gait assessments (1-4), for 4 phases of the gait cycle (DS1 =first double support phase; SS= single support phase; DS2= second double support phase; SW=swing phase; BF-RF= Biceps femoris – Rectus femoris; TA-GM= Tibialis anterior – Gastrocnemius medialis).

Discussion

Asymmetry in swing phase duration

Over the course of time, patients did not show improvements in the temporal asymmetry of the stepping pattern. Although the restoration of symmetry is one of the key objectives in the gait re-education of hemiparetic patients (e.g. Davies,

1985), as of yet, empirical data have not been able to provide unequivocal evidence for the assumption that promotion of symmetry actually leads to increased gait ability. Although some association between temporal symmetry and overall gait ability may exist (Brandstater et al. 1983), proof of a causal relationship between these two variables has not been established yet. The present results indicate that functional gait improvements may occur independent of temporal symmetrization, confirming results from studies by Hesse et al. (1993) .

The apparent independence of gait recovery and symmetrization can be understood by regarding symmetry as an emergent property of gait patterns resulting from biomechanical and neurophysiological constraints, and locomotor task demands. Following unilateral paresis, the temporal layout of the gait cycle may be optimised differently to accommodate the altered biomechanical and neurophysiological characteristics of the patient, which may result in different (i.e. asymmetrical) motor solutions (cf. Latash and Anson, 1996).

Temporal patterning of muscle (co)activity in the upper leg

Prolongation of hamstrings and quadriceps activity of the paretic leg represents a relatively common aberration in the temporal patterning of muscle activity in hemiparetic gait, in the paretic leg (Hirschberg and Nathanson, 1952; Peat et al., 1976; Knutson and Richards, 1979; Shiavi et al., 1987) as well as in the non-paretic leg. (Wortis et al., 1951; Shiavi et al., 1987). It has been argued that the massive coactivation of muscles during the stance phase of the paretic leg may be part of a primitive extensor synergy associated with the defective central control of lower extremity muscle activity following stroke (Brunnstrom, 1970; Perry, 1993). However, similar patterns of synergistic activity in the upper leg have been found in the gait of toddlers (Okamoto et al., 2003), patients with diabetic neuropathy (Kwon et al, 2003), and in patients with spinal cord injury (Leroux et al, 1999), suggesting that this abnormality represents a compensatory neuromuscular strategy rather than a primary disorder in the temporal regulation of muscle activity. During the stance phase, the BF serves primarily as a hip extensor, whereas the main function of RF during this phase is to extend the knee (Winter, 1987). Concomitant action of these muscles during the SS phase may increase the stiffness of the knee and hip joint, when the leg rotates over the foot while supporting full body weight. The compensatory coactivation of quadriceps and hamstrings muscles may be related primarily to weakness of the calf muscles, since the ankle plantarflexors have been found to supply the largest contribution to support during the SS phase

of normal gait (Kepple et al., 1997). Alternatively, increased BF- RF coactivity may be used to hold the head-arm-trunk segment against gravitational forces in case of forward postural lean (Olney and Richards, 1996).

Despite clear improvements in gait ability, the abnormal patterns of BF and RF (co-) activity that were found during the first assessment did not change towards normal. Interestingly, recent findings by Grasso et al. (2004) in spinal cord injured persons, show that restoration of the ability to step was associated with the development of prolonged BF and RF activity during the stance phase. Together with the present data, these results suggest that prolonged quadriceps and hamstring (co-) activation during stance does not form an impediment to gait recovery, but instead may reflect a rather immediate compensatory mechanism that is part of the recovery related reorganization of gait related muscle activity in neurologically impaired persons.

Temporal patterning of muscle (co)activity in the lower leg

The premature activity of the paretic GM that was found during the DS1 phase represents another characteristic timing abnormality in hemiparetic gait that has been reported by several other authors (Perry et al, 1978; Knutson and Richards, 1979; Hesse et al., 1996). Although the premature activity of calf muscles is generally associated with the presence of overactive stretch reflexes during gait, the functional implications of this hypersensitivity are still a point of debate. Several authors have questioned the contribution of spasticity to gait impairments (Berger et al., 1984; Vattanasilp et al., 2000; Ada et al., 1998; Dietz, 2003), challenging the clinical routine of reflex inhibition in mobility training after stroke. In line with this, the present data do not provide evidence that reductions in premature calf muscle activity are a prerequisite for functional gait recovery in post stroke hemiparesis. Despite substantial improvements in gait ability, the mean duration of paretic GM activity during DS1 did not change significantly over time.

General discussion

Two important questions arising from the present study is how functional improvements are possible within abnormal neuromuscular timing patterns, and why these patterns remain stable over the course of recovery. The finding that gait ability tended towards normal by using abnormal muscular timing patterns are in line with recent findings by Grasso et al (2004) in clinically complete spinal cord injured persons. They found that the stepping patterns learned during step training

were kinematic equivalents of those found in healthy subjects, despite clear differences in the patterning of leg muscle activity. Some aspects of the abnormal timing patterns found in our study (e.g. the prolonged activity of quadriceps and hamstring muscles) may be the result of a process in which neuromuscular control is optimized differently in order to obtain the best possible locomotor output in the light of impaired locomotor functioning. The present data further suggest that these optimizations may include increased coactivation levels between agonist-antagonist pairs, confirming earlier work by Lamontagne et al (2000) showing that coactivation regulation of ankle plantarflexors and dorsiflexors may be used as an adaptive mechanism in the lower leg. The present results provide a further clue that synergistic activity of muscle pairs may promote rather than impede gait performance, despite the clinical emphasis often put on the promotion of selective muscle action (Davies, 1985).

How can we account for the stability of muscle activity patterns over the course of functional gait recovery? Previous work on the muscular control of walking indicates that, within gross muscular timing patterns, local variations in force output (i.e. EMG amplitude) allow for sufficient flexibility to accommodate changes in e.g. speed or leg loading. For instance, it has been shown that for many muscles in the leg, substantial changes in gait speed can be realized by imposing phase specific adjustments in the amplitude of muscle output upon relatively invariant timing patterns (Hof et al., 2002; den Otter et al., 2004). Similarly, data are available that suggest that the amount of leg loading during walking can be decreased to a substantial degree (appr. 50%) without affecting the phasing of muscle activity despite clear changes in its amplitude (Ivanenko et al., 2002). The reason for this apparent robustness of neuromuscular timing characteristics in the context of varying task demands may be that timing and amplitude of EMG bursts during locomotion are possibly controlled differently. Whereas timing is mostly determined by central pattern generators, the amplitude depends largely on reflex loops (van de Crommert et al., 1998; Duysens et al., 2000; Dietz and Duysens, 2000).

Limitations of the study

A number of possible limitations must be taken into account with regard to the present results. It must be noted that the present findings do not rule out that other changes in neuromuscular control of the lower limbs may have occurred during gait recovery. The recovery related changes in the walking speed of patients suggest that

increasingly higher levels of muscular force output became available during recovery. Indeed, inspection of the EMG profiles recorded at these different speeds show the modulation of amplitude normally associated with speed increases (Hof et al., 2002; den Otter et al., 2004). Therefore, it may be argued that the recovery related changes in lower extremity muscle activity are primarily related to the potential force output of muscles, and that such increases in strength may be expressed as changes in the amplitude of muscle activity within relatively stable timing schemes. However, multi-session comparisons of EMG amplitude are difficult because of possible variations in electrode placement, properties of the skin tissue, and environmental conditions.

A clear limitation to this study is that the recording of EMG was restricted to 4 leg muscles that are primarily involved in gait control in the sagittal plane. Therefore, it cannot be ruled out that changes in the timing of activity occurred in other (e.g. trunk) muscle groups. Also, because the assessments period covered a period of approximately 60 days in the post-acute phase of stroke, it remains unclear whether changes in the patterning of muscle activity as measured by surface EMG might have occurred in the very early phases of recovery, or whether long term changes in patterning would have occurred.

Because this study was conducted on a treadmill, generalisation to overground walking is not straightforward. Previous studies have shown that the maximum gait speed of hemiparetic subjects on the treadmill is significantly slower and more symmetric than on a walkway (Hesse et al., 1999) and that spatiotemporal aspects of gait can be reproduced with a greater stride to stride consistency (Harris-Love, 2001). Despite these differences, there is as yet no clear evidence to show that treadmill walking affects the temporal structure of gait related muscle activity, suggesting that the present results may also be valid for overground walking. A similar point can be made with regard to the gait speeds used in this study. Because the maximum gait speed obtained during the first assessment was kept constant over all assessments, the question can be raised whether the present results allow generalization to other gait speeds. This is particularly true since the maximal obtainable gait speed of patients changed over the course of recovery. However, a longitudinal study on EMG patterns in which the maximal gait speed is used for each evaluation would be strongly confounded by gait speed.

The control group was 12 years younger than the patients so that the interpretation of the detected abnormalities is not entirely straightforward. Although little is known about the effects of age on the temporal characteristics of

muscle activity patterns, the altered coordinative strategies associated with gait in the elderly (DeVita and Hortobagyi, 2000) may result in changed temporal control of gait related muscle activity. However, because the age difference between control subjects and patients in the present study is relatively small we believe that the detected abnormalities in the data of the first assessment do not represent an age effect.

In this study, patients were allowed to hold on to a rail in case of sudden postural disturbances. It is well known that light touch of the fingertips with static objects (a so called 'haptic cue') simplifies postural control during quiet standing and walking (e.g. Jeka and Lackner, 1994). In principle, systematic changes in the frequency of handrail use may have biased our estimates of muscle (co-) activity over time. However, because the handrail was used very incidentally, and because the frequency of handrail use did not change systematically over time, we do not believe that this invalidates the present results.

Conclusion

The results of this study show that aberrant muscle timing patterns may persist even though gait improves considerably in the post-acute phase of stroke, suggesting that normalization of the temporal patterning of lower extremity muscle activity is not a prerequisite for functional gait recovery. Apparently, other neuromuscular factors than gross temporal organization, e.g. force control, may be more receptive to improvements induced by training or other interventions.

References

Adkin AL, Frank JS, Carpenter MG, and Peysar GW. Postural control is scaled to level of postural threat. *Gait Posture* 2000; 12: 87-93.

Ada L, Vattanasilp W, O'Dwyer NJ, Crosbie J. Does spasticity contribute to walking dysfunction after stroke? *J Neurol Neurosurg Psychiatry* 1998; 64:628-635.

Berger W, Horstmann G, Dietz V. Tension development and muscle activation in the leg during gait in spastic hemiparesis: independence of muscle hypertonia and exaggerated stretch reflexes. *J Neurol Neurosurg Psychiatry* 1984; 47:1029-1033.

Brunnstrom S. Motor testing procedures in hemiplegia: based on sequential recovery stages. *Phys Ther* 1966; 46: 357-375

Brunnstrom S. *Movement therapy in hemiplegia: a neurophysiological approach*. New York: Harper and Row, 1970

Brandstater ME, de Bruin H, Gowland C, Clark BM. Hemiplegic gait: analysis of temporal variables. *Arch Phys Med Rehabil* 1983; 64: 583-587.

Burridge JH, Wood DE, Taylor PN, McLellan DL. Indices to describe different muscle activation patterns, identified during treadmill walking, in people with spastic drop foot. *Med Eng Phys* 2001; 23: 427-434.

Collen FM, Wade DT, Robb GF, Bradshaw CM. The Rivermead Mobility Index: a further development of the Rivermead Motor Assessment. *Int Disabil Stud* 1991; 13:50-54.

Davies PM. *Steps to follow*. New York: Springer Verlag, 1985

De Haart M, Geurts AC, Huidekoper SC, Fasotti L, van Limbeek J. Recovery of standing balance in postacute stroke patients: a rehabilitation cohort study. *Arch Phys Med Rehabil*. 2004; 85:886-895.

DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. *J Appl Physiol*. 2000;88:1804-1811.

Den Otter AR, Geurts ACH, Mulder T, Duysens J. Speed related changes in muscle activity from normal to very slow speeds. *Gait Posture* 2004; 19: 270-278

Dietz V, Duysens J. Significance of load receptor input during locomotion: a review. *Gait Posture* 2000; 11:102-110.

Dietz V. Spastic movement disorder: what is the impact of research on clinical practice? *J Neurol Neurosurg Psychiatry*. 2003;74:820-1.

Diller L, Ben-Hishay Y, Gertsman LJ, Goodkin R, Gordon W, Weinberg MS. *Studies on cognition and rehabilitation in hemiplegia*. New York University Medical Center: Rehabilitation Monograph n. 50, 1974

Duysens J, Clarac F, Cruse H. Load-regulating mechanisms in gait and posture: comparative aspects. *Physiol Rev* 2000, 80:83-133.

Freriks B, Hermens H, Disselhorst-Klug C, Rau G. The recommendations for sensor and sensor placement procedures for surface electromyography. In: Hermens H (ed.) *European recommendations for surface elctromyography*. Enschede: Roessingh Research and Development, 1999: 15 –53

Gauthier L, Dehaut F, Joanette Y. The Bell's test: quantitative and qualitative for visual neglect. *Int J Clin Neuropsychol* 1989; 11: 49-54

Grasso R, Ivanenko YP, Zago M, Molinari M, Scivoletto G, Castellano V, Macellari V, Lacquaniti F. Distributed plasticity of locomotor pattern generators in spinal cord injured patients. *Brain* 2004; 127:1019-1034.

Harris-Love ML, Forrester LW, Macko RF, Silver KH, Smith GV. Hemiparetic gait parameters in overground versus treadmill walking. *Neurorehabil Neural Repair.* 2001;15:105-112.

Hesse S, Jahnke MT, Schreiner C, Mauritz KH. Gait symmetry and functional walking performance in hemiparetic patients prior to and after a 4-week rehabilitation programme *Gait & Posture* 1993; 1: 166-171

Hesse S, Krajnik J, Luecke D, Jahnke MT, Gregoric M, Mauritz KH. Ankle muscle activity before and after botulinum toxin therapy for lower limb extensor spasticity in chronic hemiparetic patients. *Stroke* 1996; 27: 455-460.

Hesse S, Konrad M, Uhlenbrock D. Treadmill walking with partial body weight support versus floor walking in hemiparetic subjects. *Arch Phys Med Rehabil.* 1999 Apr;80(4):421-427.

Hirschberg G G, Nathanson M. electromyogrphic recording of muscular activity in normal and spastic gaits. *Arch Phys Med Rehabil* 1952; 33: 217-225

Hof AL, Elzinga H, Grimmius W, Halbertsma JPK. Speed dependence of averaged EMG profiles in walking. *Gait Posture* 2002; 16: 78-86

Holden MK, Gill KM, Magliozzi MR, Nathan J, Piehl-Baker L. Clinical gait assessment in the neurologically impaired. Reliability and meaningfulness. *Phys Ther* 1984; 64: 35-40

Ivanenko YP, Grasso R, Macellari V & Lacquaniti F. Control of foot trajectory in human locomotion: role of ground contact forces in simulated reduced gravity. *J Neurophysiol* 2002; 87: 3070–3089.

Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients: The Copenhagen stroke study. *Arch Phys Med Rehabil* 1995; 76: 27-32

Kepple TM, Lohmann Siegel K, Stanhope S.J. Relative contributions of the lower extremity joint moments to forward progression and support during gait. *Gait Posture* 1997; 6: 1-8.

Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 1979; 120: 405-430.

Kwon OY, Minor SD, Maluf KS, Mueller MJ. Comparison of muscle activity during walking in subjects with and without diabetic neuropathy. *Gait Posture* 2003; 18:105-113.

Lamontagne A, Richards CL, Malouin F. Coactivation during gait as an adaptive behavior after stroke. *J Electromyogr Kinesiol* 2000; 10: 407-415.

Lamontagne A, Malouin F, Richards CL. Locomotor-specific measure of spasticity of plantarflexor muscles after stroke. *Arch Phys Med Rehabil* 2001; 82:1696-1704.

Latash, ML, Anson, JG What are 'normal movements' in atypical populations? *Behavioral and Brain Sciences* 1996; 19 (1): 55-106.

Leroux A, Fung J, Barbeau H. Adaptation of the walking pattern to uphill walking in normal and spinal-cord injured subjects. *Exp Brain Res* 1999, 126: 359-368

Mac Queen, J. (1967). Some methods for classification and analysis of multivariate observations. In Le Cam, L. M. and Neyman, J., editors, *Proceedings of the Fifth Berkeley Symposium on Mathematical Statistics and Probability*, volume 1, pages 281-297, Berkeley, Califonia. University of California Press.

Maki BE, Holliday PJ, and Topper AK. Fear of falling and postural performance in the elderly. *J Gerontol* 1991, 46: M123-M131.

Okamoto T, Okamoto K, Andrew PD. Electromyographic developmental changes in one individual from newborn stepping to mature walking. *Gait Posture.* 2003, 17:18-27

Olney SJ, Richards C (1996) Hemiparetic gait following stroke. Part I: Characteristics. *Gait Posture.* 1996, 4: 136-148

Peat M, Dubo HIC, Winter DA, Quanbury AO, Steinke T, Grahame MC. Electromyographic temporal analysis of gait: hemiplegic locomotion. *Arch Phys Med Rehabil* 1976; 57: 421-425

Peham C, Scheidl M, Licka T. Limb locomotion -- speed distribution as a new method for stance phase detection. *J Biomech* 1999;, 32: 1119-1124

Perry J, Waters RL, Perrin T. Electromyographic analysis of equinovarus following stroke. *Clin Orthop* 1978; 131:47-53.

Perry J. Determinants of muscle function in the spastic lower extremity. *Clin Orthop* 1993; 288: 10-26

Schenkenberg T, Bradford DC, Ajax ET. Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology* 1980; 30, 509-517

Shiavi R, Bugle HJ, Limbird T. Electromyographic gait assessment, Part 2: Preliminary assessment of hemiparetic synergy patterns. *J Rehabil Res Dev* 1987 ; 24: 24-30

Skilbeck CE, Wade DT, Hewer RL, Wood AV. Recovery after stroke. *J Neurol Neurosurg Psychiat* 1983; 46: 5-8

Van de Crommert HW, Mulder T, Duysens J. Neural control of locomotion: sensory control of the central pattern generator and its relation to treadmill training. *Gait Posture* 1998; 7:251-263.

Vattanasilp W, Ada L, Crosbie J. Contribution of thixotropy, spasticity, and contracture to ankle stiffness after stroke. *J Neurol Neurosurg Psychiatry* 2000; 69: 34-39.

Wade DT, Hewer RL. Functional abilities after stroke: measurement, natural history and prognosis. *J Neurol Neurosurg Psych* 1987; 50: 177-182

Wade DT. *Measurement in neurological rehabilitation*. Oxford: Oxford Medical publishers, 1992

Wilson B, Cockburn J, Halligan P. *Behavioral Inattention Test*. Titchfield, Fareham, Hants, England: Thames valley Test Co.; Gaylford MI: National Rehabilitation services, 1987

Winter DA. The biomechanics and motor control of human gait. Waterloo, Ontario: University of Waterloo Press, 1987.

Wortis SB, Marks M, Hirschberg GG, Nathanson M. Gait analysis in hemiplegia. *Trans Am Neurol Assoc* 1951; 76: 181-183